Answer to Dermacase *continued from page 247*

4. Favre-Racouchot syndrome

Favre-Racouchot syndrome (FRS), first noted by Favre in 1932,[1] and detailed by Favre and Racouchot in 1951,[2] is a condition characterized by cysts, comedones, and nodular elastosis in sun-damaged skin. It most commonly presents in elderly white men with a history of long-term sun exposure and heavy smoking.[3,4] Favre-Racouchot syndrome is a disease of cosmetic concern, limited to the skin with no related internal manifestations.

Epidemiology

Favre-Racouchot syndrome is not an uncommon condition—it occurs in up to 6% of those older than 50 years of age.[5] The incidence of FRS increases with age, although it has been reported in patients as early as in their second decade of life.[6] Prevalence is highest in white men, but the condition has been observed in women and dark-skinned individuals.[7,8] Although the exact mechanism of the condition is not known, FRS has been specifically connected to sun exposure,[9] smoking,[3] and, in a minority of cases, radiation exposure.[10]

Etiology and pathogenesis

Favre-Racouchot syndrome is characterized by solar elastosis, with the presence of nodules, cysts, and comedones. Solar elastosis refers to the damage to dermal elastic tissue due to prolonged exposure to UVA and UVB rays. The histologic changes present in FRS include an atrophic epidermis and large masses of keratinous material, causing follicular plugging, as well as solar elastotic changes (which include increased elastic tissue with thickened, tortuous fibres in the upper and mid dermis) and possible basophilic degeneration of connective tissue.[8] Although it is suggested that senile comedones and sebaceous gland hyperplasia are somehow associated with photodamage, no proof of this has been documented.[11] Interestingly, the comedones found in FRS are histologically indistinguishable from the primary comedones of acne vulgaris, with the exception of a lack of inflammation and the presence of a marked actinic elastosis in the surrounding dermis.[10,12] It is hypothesized that damage to the dermal elastic network and subsequent reduction of tensile strength might be what predisposes these comedones to histologic change, expansion, and subsequent formation of cysts and nodules.[6,8]

Diagnosis and differential

Favre-Racouchot syndrome can be diagnosed by clinical findings of multiple open and closed comedones with yellowish nodules of elastotic material in patients older than 40 years of age. Patients with FRS usually present with a history of long-term sun exposure and smoking, and sometimes the possibility of radiation exposure, although this is rare. Clinically, examination reveals histologic changes manifested as actinically damaged skin with atrophy; yellowish discolouration; wrinkles and furrows; cystic nodules; and punctate, waxy, noninflamed, soft, open or closed comedones.[4,8] As it is hypothesized that the underlying etiology of FRS is UV damage, lesions will generally be photodistributed. Affected areas of the body are primarily the periorbital and temporal areas, but the malar eminences, lateral neck, earlobes, and postauricular areas might also be involved.[4] The eruptions are usually bilateral and symmetric but can be asymmetric or unilateral, depending on the history of sun exposure.[13] As a member of the spectrum of skin conditions caused by long-term sun exposure known as dermatoheliosis, FRS can be associated with actinic keratoses, basal and squamous cell carcinoma, cutis rhomboidalis nuchae, trichostasis spinulosa, and keratoacanthoma.

The differential diagnosis for FRS includes dermatoses that present with lesions of similar morphologies, but it can nonetheless be distinguished based on a variety of features. These features include the following: acne vulgaris (lesions limited to inflamed comedones); sebaceous hyperplasia (lesions limited to enlarged sebaceous glands); actinic comedonal plaques (similar lesions, also found on the flexural surface of the arms and often unilateral); cutis rhomboidalis nuchae (similar skin morphology, differentiated by the presence of deep, crisscross furrows on the dorsal and lateral skin of the neck); chloracne (similar lesions, but also localized in postauricular areas, axillae, and groin); actinic granuloma (different lesional morphology: pink or skin-coloured papules in annular or serpiginous plaques with atrophic centres); colloid milium (different lesional morphology: small, amber, waxy, translucent, and firm papules occurring in groups); and syringomas or trichoepitheliomas (both presenting with similar lesions but occurring in a younger patient population compared with FRS).[4,14]

Treatment

It is important to note that, unless treated, FRS is slowly progressive. First, it is key that all patients are advised to take proper sun precautions, such as wearing sunscreen with a sun protective factor of at least 30 with UVA and UVB protection and avoiding the outdoors between 10 AM and 2 PM if at all possible. If the patient is a smoker, quitting is strongly advised. In treating the patient, 2 different approaches can be used: pharmacologic and surgical. Topical retinoids, such as tretinoin, adapalene, or tazarotene, are the most effective pharmacologic treatments. Tretinoin, in addition to eliminating comedones, has been shown to reduce the aged appearance of sun-damaged skin and has beneficial effects on collagen.[15] Retinaldehyde, a precursor to retinoic acid, shows similar effectiveness; in addition, it has been shown to increase the surface area of elastin and

collagen fibres in UVA-exposed skin.[16] Surgical techniques include excision, dermabrasion, curettage, comedone extraction, and laser resurfacing. Although these techniques have yielded poor results when used independently, they allow FRS a very favourable outcome when used in conjunction with medication.[4,14]

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Competing interests
None declared

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